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Perceptual correlates of state-dependent learning

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ABSTRACT

Perceptual Correlates of State-Dependent Learning

Sixty food-deprived rats were placed in a tone (10,000 Hz.) no-tone discrimination (which was used to generate generalization gradients in extinction). Half of the Ss received a 50 mg./kg. injection of Atropine Sulfate (interperitoneal (I.P.) while half received the same dose of Saline (I.P.). After seven days of discrimination training the two acquisition groups (atropine and saline) were again divided into the same drug and non-drug groups. There were, therefore, four acquisition-extinction drug combinations referred to as: Drug-Drug (D-D), Non-Drug - Non-Drug (ND-ND), Non-Drug - Drug (ND-D), and Drug - Non-Drug (D-ND). In extinction (which lasted five days) an S was extinguished at one of five frequency values: 10,000; 8,000; 7,000; 5,000; and 3,000 Hz. Four generalization functions, one for each acquisition-extinction drug combination, were of primary interest. Interpretations of the generalization gradients were confounded by the finding that atropine interfered with the acquisition of discrimination. However, it was concluded that atropine did not interfere with learning variables but affected only performance variables. While dissociation or state-dependent learning was reported for both state change groups, a much fattened generalization function was reported for the ND-D group. It was concluded that two mechanisms differentially mediate state-dependent learning (ND-D dissociation mediated by perceptual changes, D-ND dissociation mediated by the drug acting as a discriminative stimulus). It was further concluded that this dual mechanism view of state-dependent learning is consistent with the phenomenon of asymmetrical dissociation discussed by Overton (1968).

PERCEPTUAL CORRELATES OF STATE-DEPENDENT LEARNING

by

Philip Slonim

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

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Perceptual Correlates of State-Dependent Learning

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INTRODUCTION

The phenomenon of state-dependent or dissociated learning was first demonstrated by Girden and Culler (1937). Studying leg flexion in dogs, they found that if a dog had been conditioned while not drugged, it would subsequently fail to respond under curare. They report that failure to respond was not due simply to paralysis, for a response to the unconditioned stimulus could still be observed. In addition, a dog conditioned while drugged with curare would subsequently respond when the drug state was reinstated, but would not respond while non-drugged. Girden and Culler concluded that the responses performed by the non-drugged animal were somehow separated from that performed by the same animal when drugged. Dissociation of learning, as they called the phenomenon, referred to this absence of transfer between the drugged and non-drugged state.

State-dependent learning, aside from being an area of theoretical interest, has some important experimental application. As Overton states (1964), experimentors attempting to study the effects of drugs on behavior must apparently design their experiments so as to separate out the effects of a particular drug per se from the effects to changes in the drug state during the experiment. Another area in which the dissociation of learning appears highly important is drug treatment in the clinical setting. An implicit assumption at any such treatment is that therapeutic gains made under a drug generalizes or transfers to the normal non-drug state. However, a paper by Barry, Etheridge, and Miller (1965), indicates that this assumption may be unwarranted. Rats which underwent counter-conditioning and extinction of fear and avoidance under the drugged state (10, 30, 30 mg./kg. of sodium amobarbital) failed to transfer to the non-drugged state. This non-transfer for the three

amobarbital dosage groups was measured by a decrease in the percentage of animals who pressed a lever from the last drugged trial to the first non-drugged trial. The possibility, therefore, exists that any gains accrued from therapeutic treatment with the aid of a change of state (drugs, hypnosis, etc.) will entirely or in part fail to transfer to the Ss non-drugged state. Apparent gains under such circumstances might be only temporary and appear only in the state in which they were acquired.

Overton (1964) performed a series of experiments which renewed interest in the above effect. Rats were trained to turn right or left in a T-maze to escape shock. Overton reports that animals who learned to escape shock by turning right in the drugged condition (sodium pentobarbital) would, upon being tested without the drug, turn left or right at chance level. However, for animals who learned and were tested under identical conditions or states right or left, responding was significantly above chance level. Complete dissociation was produced by heavy doses of sodium pentobarbital while partial dissociation was found at lower doses. As Overton concludes, the more similar two drug states are, the more total the transfer of training between them. Overton (1964) considers the possibility that the ability of drug state changes to produce performance decrements may be based on sensory cue changes of internal discriminative stimuli; yet he has been unable to mimic the effects of drug state changes with selected extreoceptive and interoceptive sensory changes.

The phenomenon of dissociation has been extended for several classes of drugs and varied general experimental techniques. Several of the latter are: escape learning (Overton, 1964), successive discrimination reversal (Bindra and Reichert, 1967), and passive avoidance (Otis, 1964). Otis, for example, trained rats in a passive avoidance response while under the influence of chlorpromazine and then tested the Ss after they received an injection of

saline or another 1.25 mg./kg. of chlorpromazine. Animals that learned the task under one condition and tested in the other condition showed, according to Otis, greater dissociation and less recovery than animals which received only injections of saline or chlorpromazine during training and testing sessions. In a somewhat different approach Bindfa and Reichert (1967) adopted a successive discrimination reversal task using escape from shock in a T-maze. After a habit, turning left or right to escape a shock under drug or non-drug conditions was well established, both drug state and habit were reversed. As hypothesized, the effect of a change in drug state from acquisition to reversal facilitates reversal indicating, according to the authors, dissociation manifested by non-transfer from drug to the non-drug state.

Considering the techniques available for studying state-dependent learning, the differential response technique seems most promising (Overton, 1966). Utilizing this method in a T-maze with escape from shock rats were trained to turn right under one state and left in the presence of another state. Overton reports that the rate of acquisition of the differential response is dose and drug type dependent. It, then, follows that the slower appearance of differential responses indicates partial dissociation between two states while rapid acquisition indicates more complete dissociation. Overton also found a number of categories of drugs which produce dissociation. These are: anesthetics, tranquilizers, antimuscarinics (i.e., Atropine), and convulsants. Because of the wide range of drugs which produced dissociation Overton concluded that not all dissociation phenomenon are the result of a single unidimensional process.

With Overton's adoption of the differential response technique, there would appear to be an implicit assumption that by simplifying the stimulus

space, the dissociated learning phenomenon might be better understood (stimulus space referring to total stimulation impinging on the organism). This trend to simplify the stimulus space is perhaps more clearly seen in a somewhat later paper (Overton, 1969). Again utilizing the differential response technique with an escape procedure, Overton reports that if rats were blinded before training, they would still be able to learn drug-controlled differential responses. Overton also reports that when differential shock conditions were provided as cues, response control did not appear as rapidly, indicating to Overton that response control exercised by pentobarbital is not mediated by altered perception of the shock (analgesia) nor to any great degree by altered visual perception. However, it should be pointed out that the fact that blinded rats can still acquire differential responses does not exclude the role of vision in the normal, non-blinded rat.

While the above trend to decrease complexity of the stimulus space has certain benefits, there are some inherent difficulties. The procedure, of course, allows the E to observe the effects of state changes upon the "physiological substratum of the organism" while limiting, to a degree, confounding external stimulus inputs. The logical conclusion of such a program, it seems, is to totally deafferent an experimental S and observe whether it is possible to establish differential responding to various imposed states utilizing either some behavioral or some physiological measure. Aside from the obvious technical difficulty of such a program, state change decrements observed in such a setting would be most difficult to interpret: "...As maze learning ability is decreased by deafferentation, the decrement could also occur without any decrease in drug state distinctiveness." (pg. 20, Overton, 1969). As Overton concludes "... unless some particular sensory system is responsible for drug state discrimina-

tion, it may be impossible to demonstrate that the sensory systems are involved, even if they are." (pg. 20, Overton, 1969). The present study will attempt to investigate the phenomenon of state-dependent or dissociated learning from a different perspective than the above mentioned studies. More specifically, the above mentioned trend in state-dependent research will be abandoned for a more complex, but hopefully, more informative stimulus generalization paradigm. Barry, Etheridge, and Miller (1965) state, "It is a well known fact that many drugs produce novel sensations and other changes in the stimulus situation. Therefore, learning which has occurred in the drug state may be expected to suffer a stimulus generalization decrement in transferring to the non-drug state." (pg. 155) The possibility, therefore, exists that the state changes in fact effect stimulus generalization gradients in an orderly and predictive manner. If state changes effect generalization gradients in such a manner, it may be argued that the mediating factor for state-dependent or dissociated learning is perceptual in nature.

Stimulus generalization gradients are thus viewed as a measure of the functioning perceptual system. It is, therefore, hypothesized that state-dependent learning as established by differential drug states will effect generalization gradients in an orderly and predictive manner. More specifically, if the differential drug state produces a situation of no transfer from drug to non-drug states, no generalization gradient should be observed since the stimuli should now appear less similar to the original CS due to a shift in the stimulus continuum from which the stimulus generalization gradient is generated. However, if there is only partial dissociation produced by the drug to non-drug (D-ND) and non-drug to drug (ND-D) shift, a gradient should still be observed. Its shape should, however, be somewhat flattened indicating that the stimuli appear less similar to the original CS. Where there is no state

change as in drug to drug (D-D) and non-drug to non-drug (ND-ND) conditions, the same stimulus generalization functions should appear.

METHOD

Subjects. Sixty male albino Sprague-Dawley rats supplied by Huntington Farm Distributors served as Ss. They were 60 to 90 days old and weighed approximately 200 grams upon their arrival. The Ss were housed individually in their home cages.

Apparatus. A Scientific Prototype Skinner Box served as the experimental chamber. The box was altered in two ways: the water bottle was moved next to the food well, and a horn type tweeter was mounted onto the plexiglass top of the box. The speaker was placed so that one end was over the bar while its far end reached the wall opposite the bar. Driving the tweeter was an Audio Generator model number 1304-B. Lehigh Valley Programming equipment was utilized in establishing the desired experimental situation which will be elaborated in the following section. The frequency setting on the generator was set on normal and the output was set at 7 volts. It should be noted that the tweeter was capable of generating pure tones to frequencies up to approximately 17,000 Hz.

During the experiment the frequency values used were 10,000, 8,000, 7,000, 5,000, and 3,000 Hz. The sound pressure at each of these frequency levels was as follows: 90, 98, 98, 92, and 98 decibels respectively. These values were calculated with a General Radio Sound Level Meter model #1551-C.

The apparatus was located in an experimental cubicle approximately 7 x 4 feet. The only illumination during an experimental session was a low illumination light above the bar. Reinforcement pellets were Noyes Precision Food Pellets (45 mg.).

The drug utilized was Atropine Sulfate in powder form supplied by a local pharmacy. Physiological saline was used to mix the drug and was also used

as the control injection (injections were given interperitoneally (I.P.) with a 24-gauge needle). Separate syringes were used for the I.P. and control injection.

Procedure. The following will describe the treatment of the Ss upon their receipt from the distributors. This description applies for all three replications except when otherwise indicated. Five days after the Ss arrival, they were placed on an ad lib feeding schedule which lasted for one week. It should be noted that water was always available in the Ss home cage. Following this period the Ss weight was gradually decreased by 20% of his average ad lib weight. On the day following the S reaching this new weight level, he was pretrained to bar press utilizing usual operant training techniques. On the day following the successful completion of the 100 bar presses, the S was randomly assigned to one of twenty experimental groups /See Table 1/ (using the table of random numbers). The experiment proper, then, began for that S.

The experiment proper consisted of an auditory discrimination task. The S^D was the presence of a 10,000 Hz. tone, the S^Δ was the absence of the tone. Discrimination training lasted seven days and consisted of 30 trials a day, each lasting one minute. There were always 16 S^D periods and 14 S^Δ periods. The order of presentation of these periods was randomized every other day with the restriction that no more than 3 S^D or 3 S^Δ periods could occur consecutively. In addition, for all Ss a day of training always began and ended with an S^D period. It should be noted that the tone during the S^D period lasted for an entire minute and the S was on a continuous reinforcement schedule during that period. In acquisition the S received either a daily 50 mg./kg. I.P. injection of Atropine Sulfate or an I.P. injection of isotonic saline (this dosage is same used by Overton). The body weight to dosage ratio was 1 cc./kg.

The injection (whether Atropine or Saline) was given 15 minutes prior to the beginning of the session, this time was to insure that the drug had taken effect before the session was initiated. Following the seven days of acquisition training, the extinction trials began. In extinction, an S was either in a drugged state or non-drugged state with the dosage levels the same as during acquisition. The Ss in extinction were also in one of the following five acquisition frequency groups: 10,000, 8,000, 7,000, 5,000, or 3,000 Hz.

In summary, then, there were 20 experimental groups; the experimental design is summarized in Table 1. The experiment consisted of three replications. In the first replication, all 20 Ss were run simultaneously, each one assigned to 1 to 20 experimental groups. For the second replication, the D-D and D-ND groups were run first followed 13 days later by the ND-ND and ND-D groups, and for the final replication the order of the second group was reversed. This counterbalancing was carried out to enable the E to compare the D-D to the D-ND group, and the ND-ND to the ND-D group with running order controlled. There were in all 12 Ss from the 3 replications who died before completing the experiment. They came from the following groups: 3 from ND-D, 4 from D-ND, and 5 from D-D. Death appeared to be the result of respiratory failure. These Ss were replaced by adding 12 Ss at the completion of the third replication.

TABLE 1

EXPERIMENTAL DESIGN

Acquisition		Extinction ND					Extinction D				
		1	2	3	4	5	1	2	3	4	5
ND	10,000										
	8,000										
	7,000										
	5,000										
	3,000										
D	10,000										
	8,000										
	7,000										
	5,000										
	3,000										

RESULTS

The grouped acquisition and extinction data, although not of primary interest in the present study, is presented in Figures 1 and 2. The two figures represent the acquisition and extinction curves for the four experimental groups. It is clear that for the above figures the acquisition functions for the drugged and non-drugged Ss are different. For Figure 1 (S^D responses) the non-drugged Ss respond with more correct responses per day than the drugged Ss. Figure 2, representing the S^Δ responses per day of acquisition and extinction, clearly shows the inverse function of Figure 1 - that is, drugged Ss respond incorrectly more often than non-drugged Ss as acquisition proceeds. It should also be noted that for the extinction functions the differences between the drugged and non-drugged groups are not as great over the last three days as for the acquisition functions represented in Figures 1 and 2.

Since the central concern of this study was the generalization functions, hence extinction, two dependent measures from extinction are of greatest interest. These two measures were chosen since they allow each S's acquisition responses to be used as a base measure to calculate a dependent measure ratio. Table 2 presents the results of the analysis of variance on measure 1 or the ratio of the S^D responses per day of extinction to the total number of S^D responses in acquisition. Table 3 presents the analysis of variance on measure 2, or the ratio of S^Δ responses per day of extinction to the total number of S^Δ responses during acquisition.

Figure 1. S^D responses per day of acquisition and extinction for all Ss.

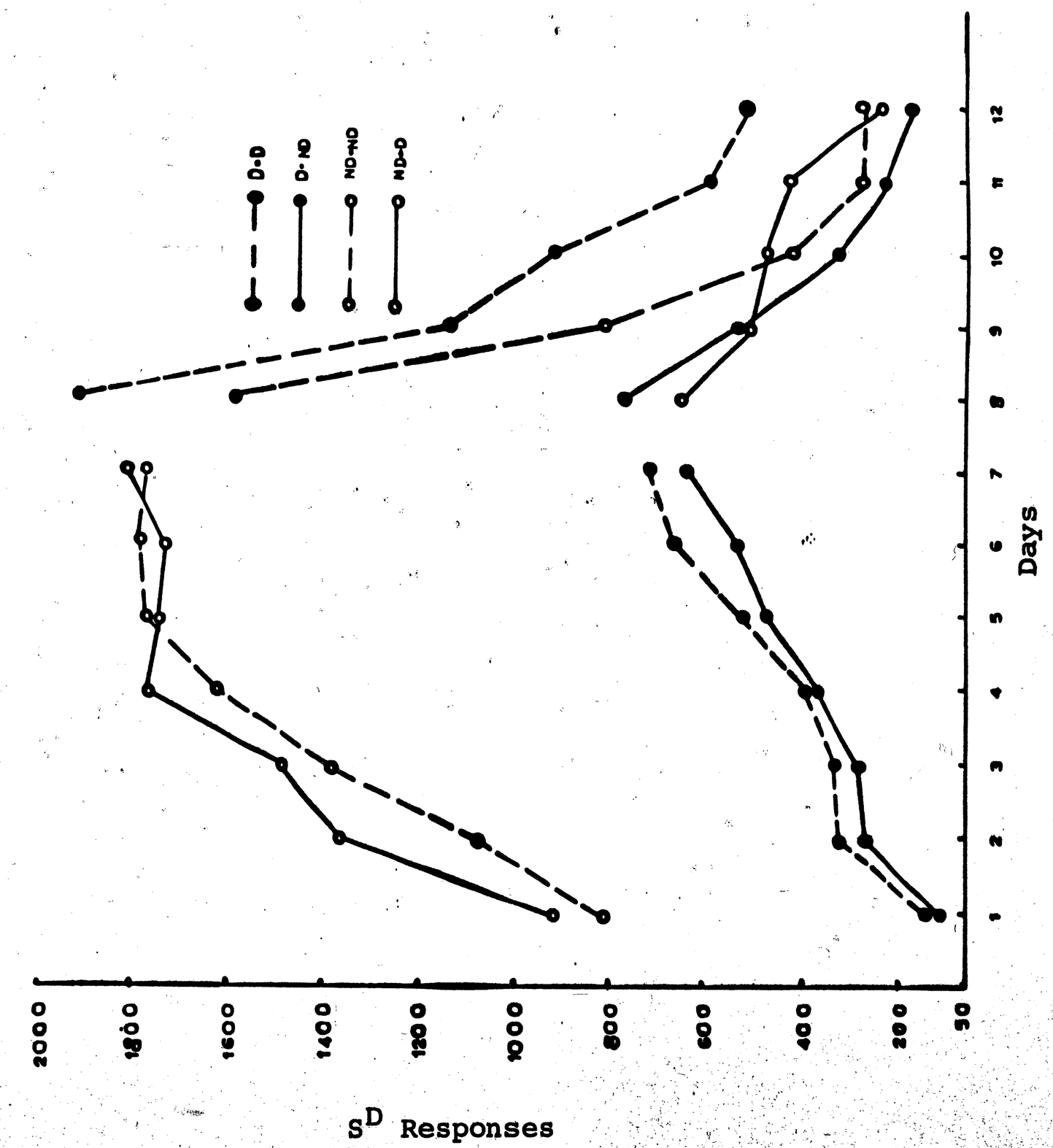

D-D
D-ND
ND-ND
ND-D
8
9
10
11
12
1
2
3
4
5
6
7
Days
2000
1800
1600
1400
1200
1000
800
600
400
200
50
S^D Responses

Figure 2. S^{Δ} responses per day of acquisition and extinction for all Ss.

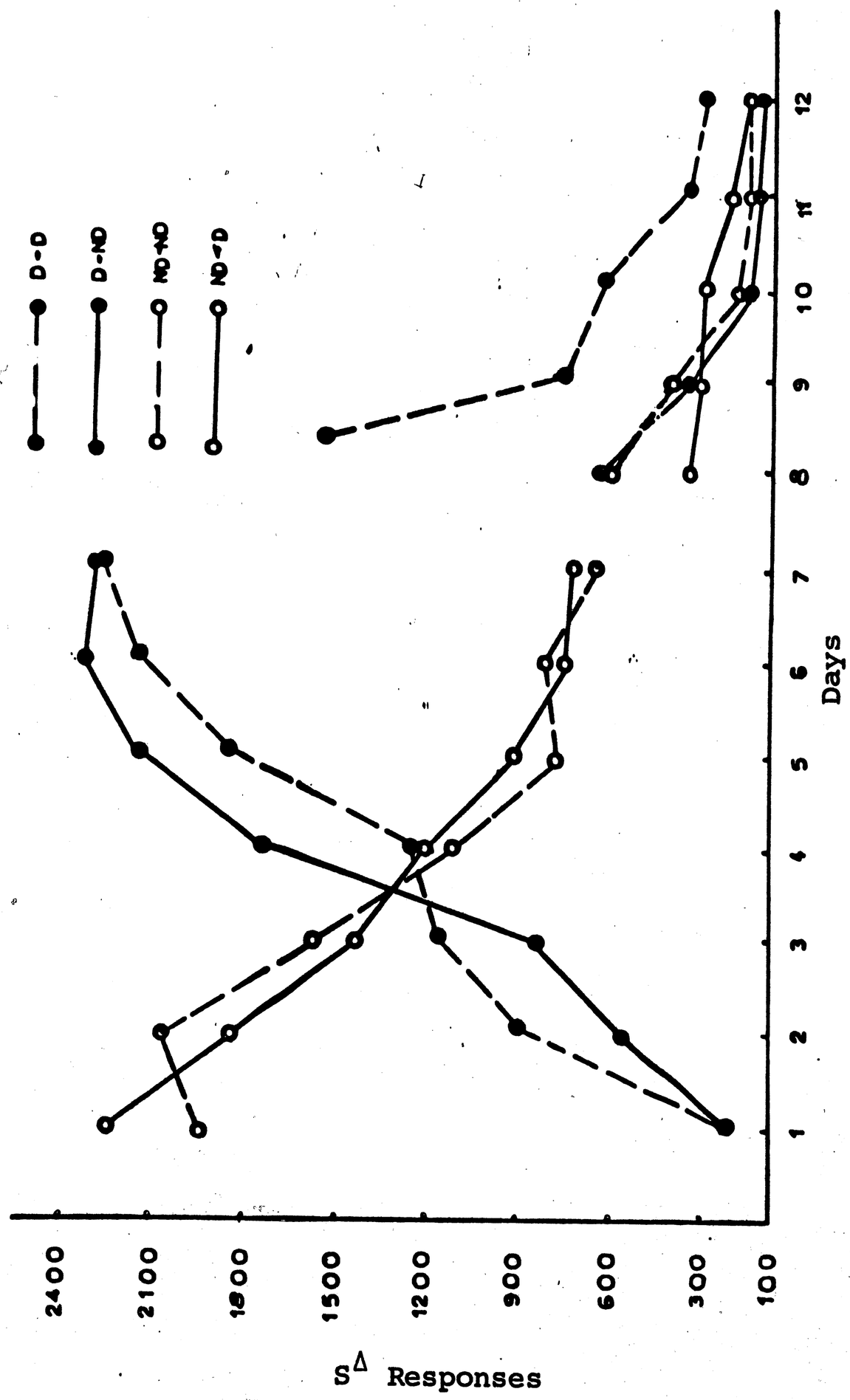

0-0
0-2
2-2
2-0
12
11
10
9
8
7
6
5
4
3
2
1
Days
2400
2100
1800
1500
1200
900
600
300
100
S^Δ Responses

Table 2

Analysis of Variance for the

SD extinction per day/ SD acquisition (total) measure

SOURCE	SS	DF	MSQ	F
Acquisition(A)	4.537027	1	4.537027	231.73**
Extinction (E)	.8942388	1	.8942388	29.85**
Frequency (F)	2.918298	4	.7295744	57.17**
Days (D)	.7000648	4	.1750102	7.41**
Replication (R)	.03660503	2	.01830251	- - -
AE	1.335895	1	.1335895	30.48**
AF	1.307754	4	.3269385	30.44**
AD	.4999099	4	.1249775	7.11**
AR	.03914937	2	.01957468	- - -
EF	.1048678	4	.02621695	4.73**
ED	.147495	4	.03686409	2.29
ER	.05990047	2	.02995023	- - -
FD	.8024348	16	.05015217	3.77**
FR	.1020838	8	.01276048	- - -
DR	.1890603	8	.02363253	- - -
AEF	.4341232	4	.1085308	8.84**
AED	.263644	4	.06591111	3.21
AER	.08765302	2	.04382651	- - -
AFD	.7242653	16	.04526658	4.73**
AFR	.08589554	8	.01073694	- - -
ADR	.1405695	8	.01757119	- - -
EFD	.5082643	16	.03176652	2.15*

Table 2 (continued)

SOURCE	SS	DF	MSQ	F
EFR	.04425277	8	.005531596	- - -
EDR	.1289182	8	.01611491	- - -
FDR	.4250534	32	.01328292	- - -
AEFD	.5212811	16	.03250007	1.81
AEFR	.09821735	8	.01227717	- - -
AEDR	.1643726	8	.02054657	- - -
AFDR	.3064081	32	.009575252	- - -
EFDR	.4717921	32	.01474350	- - -
AEFDR	.5757517	32	.01797224	- - -

* (p. < .05)

** (p. < .01)

Table 3

Analysis of Variance for the

S^{Δ} extinction per day/ S^{Δ} acquisition (total)measure

SOURCE	SS	DF	MSQ	F
Acquisition (A)	.07496786	1	.07496786	8.34
Extinction (E)	.01383666	1	.01383666	2.11
Frequency (F)	.1765960	4	.04414901	30.89**
Days (D)	.04961405	4	.01240351	1.15
Replication (R)	.009085442	2	.004542721	- - -
AE	.01742847	1	.01742847	1.71
AF	.04252961	4	.01063240	4.85*
AD	.04315365	4	.01078841	1.61
AR	.01795853	2	.008979265	- - -
EF	.004407464	4	.001101866	1.00
ED	.03784393	4	.009460983	.91
ER	.01311873	2	.006559365	- - -
FD	.04942829	16	.003089268	1.44
FR	.01140215	8	.001425269	- - -
DR	.08609322	8	.01076165	- - -
AEF	.01922938	4	.004807345	5.47*
AED	.03168312	4	.007915780	1.84
AER	.02041928	2	.01020964	- - -
AFD	.04813476	16	.003008422	2.01*
AFR	.01753872	8	.002192341	- - -
ADR	.05357795	8	.006697244	- - -
EFD	.01646287	16	.001028929	- - -

Table 3 (continued)

SOURCE	SS	DF	MSQ	F
EFR	.008529594	8	.001066199	.40
EDR	.09084668	8	.01135583	- - -
FDR	.06882857	32	.02150893	- - -
AEFD	.01708898	16	.001068061	.53
AEFR	.007028249	8	.0008785311	- - -
AEDR	.03433081	8	.004291351	- - -
AFDR	.04795982	32	.001488744	- - -
EFDR	.08292192	32	.002591310	- - -
AEFDR	.06510688	32	.002034590	- - -

*(p. < .05)

**(p. < .01)

Since this study was concerned with comparing drug state changes and their effect on generalization functions, only the following sources of variance will be discussed: the acquisition x extinction interaction, the acquisition x extinction x frequency interaction, and the acquisition x extinction x frequency x day interaction. The 2-way interaction was significant at the .05 level for the S^D dependent measure ratio, significance was not reached at the .05 level for the S dependent measure ratio. The 3-way interaction was always significant at the .05 level while the 4-way interaction never reached significance at the .05 level. Figure 3 presents the significant acquisition x extinction interaction. This figure indicates that the state change groups responded less than the appropriate non-changed group (D-ND group responded less than D-D group and the ND-D group responded less than the ND-ND group). Figures 4 and 5 present the acquisition x extinction x frequency interaction for the two dependent measures, each figure represents the generalization gradients for each of the four acquisition-extinction drug combinations, utilizing one of the two dependent measures. It should be noted that for both Figures 4 and 5, generalization gradients appear for only three of the four acquisition-extinction drug combinations, the ND-D gradient appears flattened while the remaining three (ND-ND, D-ND, and D-D) appear normal, that is, non-flattened.

Figure 3. A x E interaction for the S^D extinction per day/ S^D acquisition (total)measure

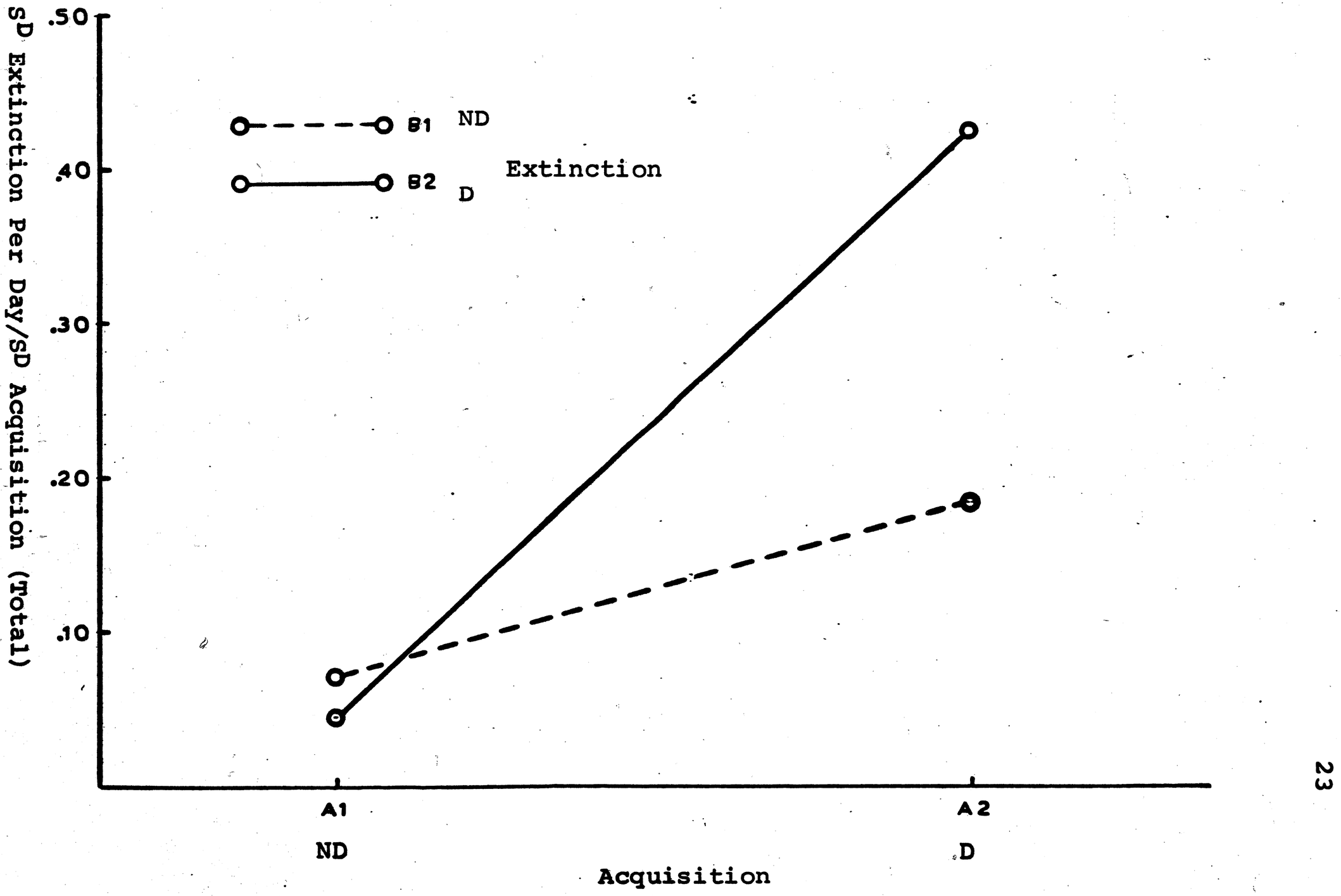

SD Extinction per Day/SD Acquisition (Total)
.50
.40
.30
.20
.10
B1 ND
B2 D
Extinction
A1
ND
A2
D
Acquisition

Figure 4. A x E x F interaction for the S^D extinction per day/ S^D acquisition (total) measure

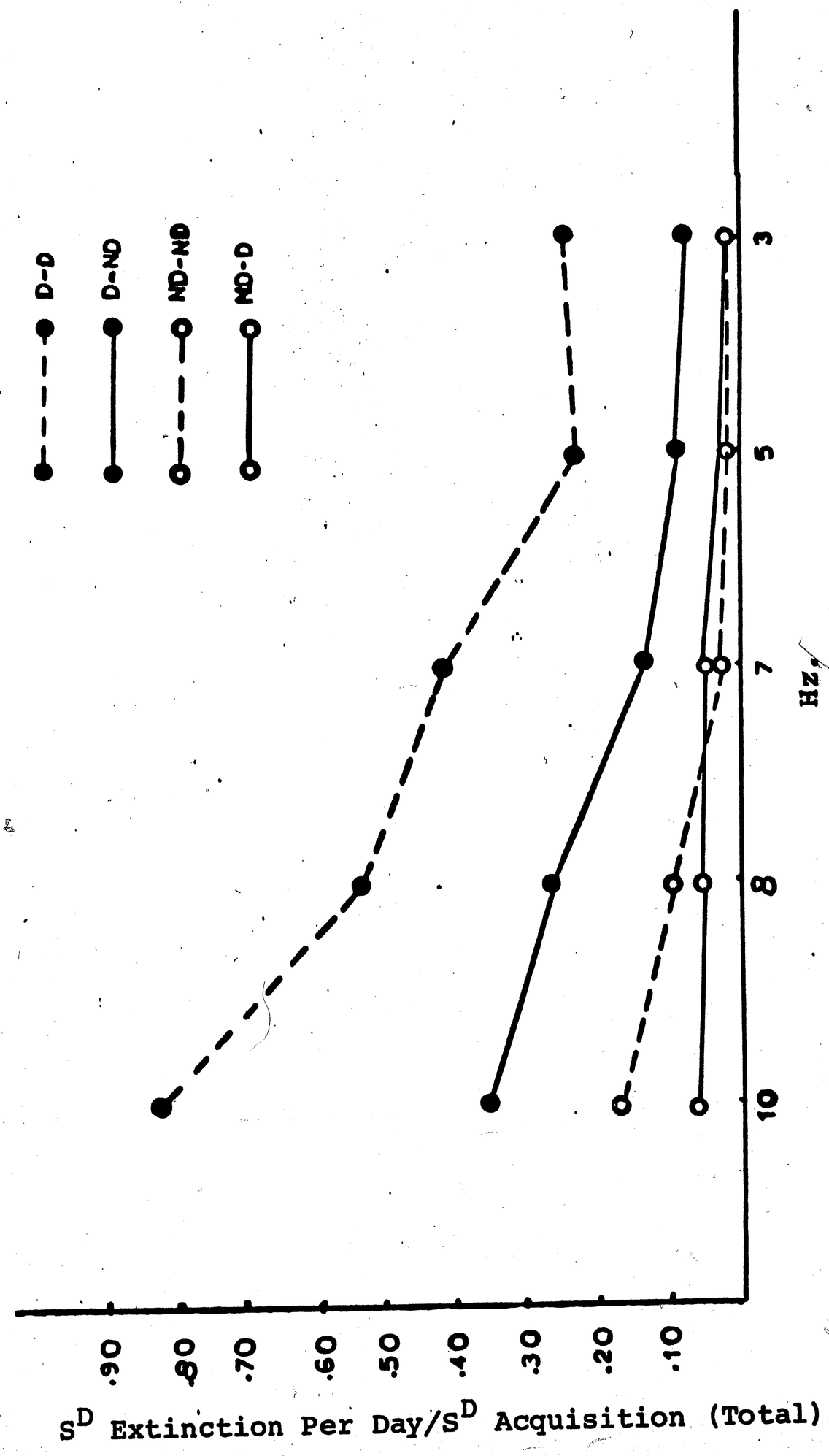
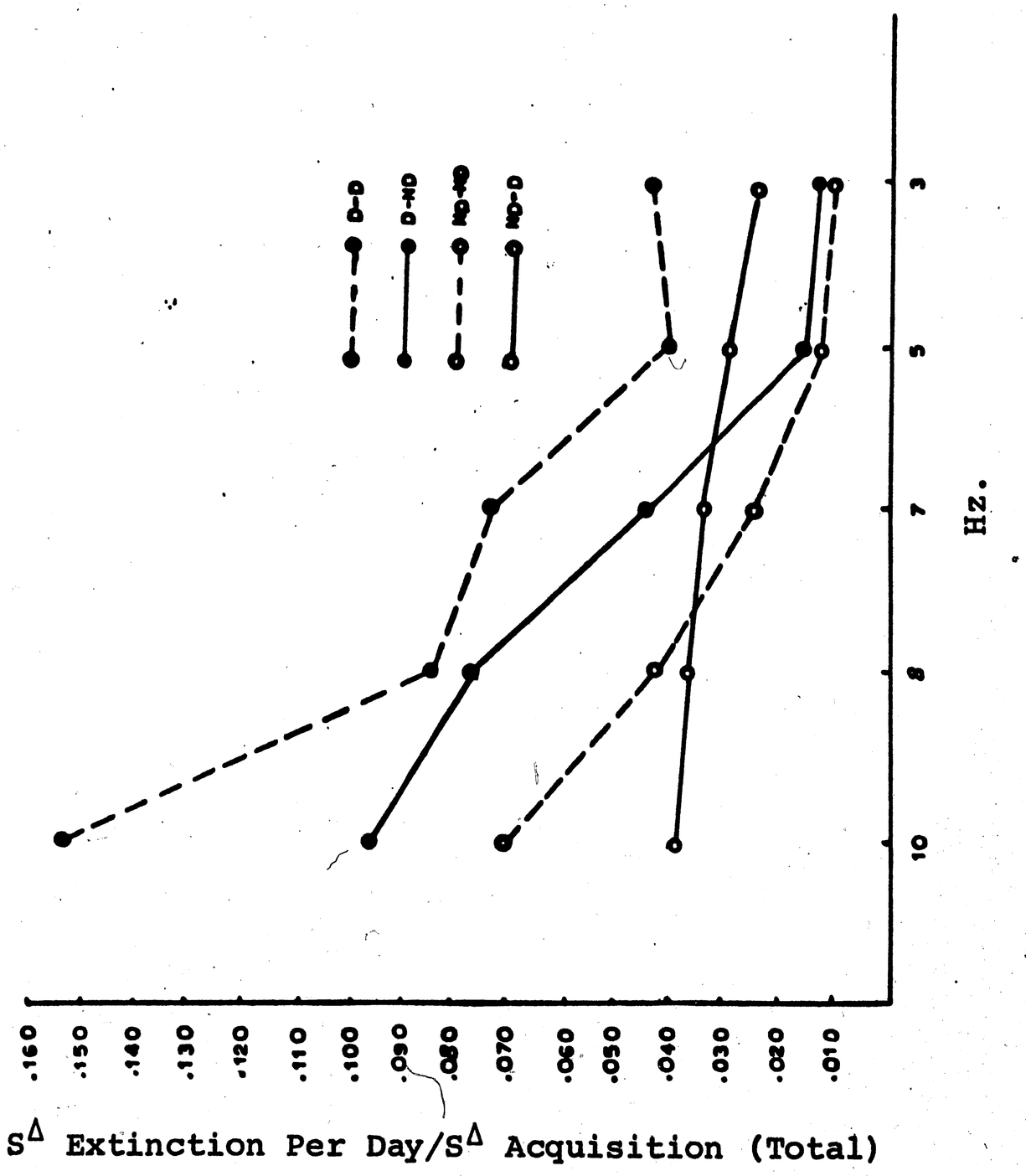

D-D
D-ND
ND-ND
ND-D
3
5
7
8
10
Hz.
.90
.80
.70
.60
.50
.40
.30
.20
.10
S^D Extinction Per Day/S^D Acquisition (Total)

Figure 5. A x E x F interaction for the S^{Δ} extinction per day/ S^{Δ} acquisition (total) measure


D-D
D-ND
ND-D
ND-ND
3
5
7
8
10
Hz.
.160
.150
.140
.130
.120
.110
.100
.090
.080
.070
.060
.050
.040
.030
.020
.010
S^Δ Extinction Per Day/S^Δ Acquisition (Total)

DISCUSSION

The difference between the acquisition functions for the non-drugged (ND) and drugged (D) Ss cannot be explained by a simple analysis of Figures 1 and 2. There are several mechanisms which could account for these functions; however, they are not mutually exclusive and will be offered as tentative explanations only. Hines, Miller and Lee (1969)[1] have shown that atropine sulfate in dosages as small as 2 mg./kg. (the present study used 50 mg./kg.) can suppress food reinforced responding. The suppression, they conclude, is due to peripheral and not central factors since atropine does not appear to pass through the blood-brain barrier easily. It is, therefore, probable that the dosage levels used in this study had the peripheral effect of making the S more thirsty, thus making the food less reinforcing by lowering the S's incentive motivation - the overall effect being a suppression of S^D responses in acquisition. While this explanation is consistent with Figure 1, it alone cannot explain Figure 2 which indicates an increase in S^Δ responding for the D Ss as acquisition proceeds. One explanation for this increase is that since S^D responding was effectively suppressed by atropine, learning (possibly indicated by an increase in S^D responding) could only be effectively expressed by an increase in S responding. S^Δ responding can, thus, be viewed as simply a displacement of suppressed learning which would in a normal ND state be expressed by a greater increase in S^D responses. Another possible view which could account for the above-mentioned increase in S^Δ acquisition responding under atropine, is that atropine itself has some effect on this specific response measure. Bivens and Ray (1968) have shown that relatively low dosages of atropine (3, 6, 9, and 12 mg./kg.) can act to disrupt inhibition of non-rewarded (S^Δ) responses. While the former view is favored at this time,

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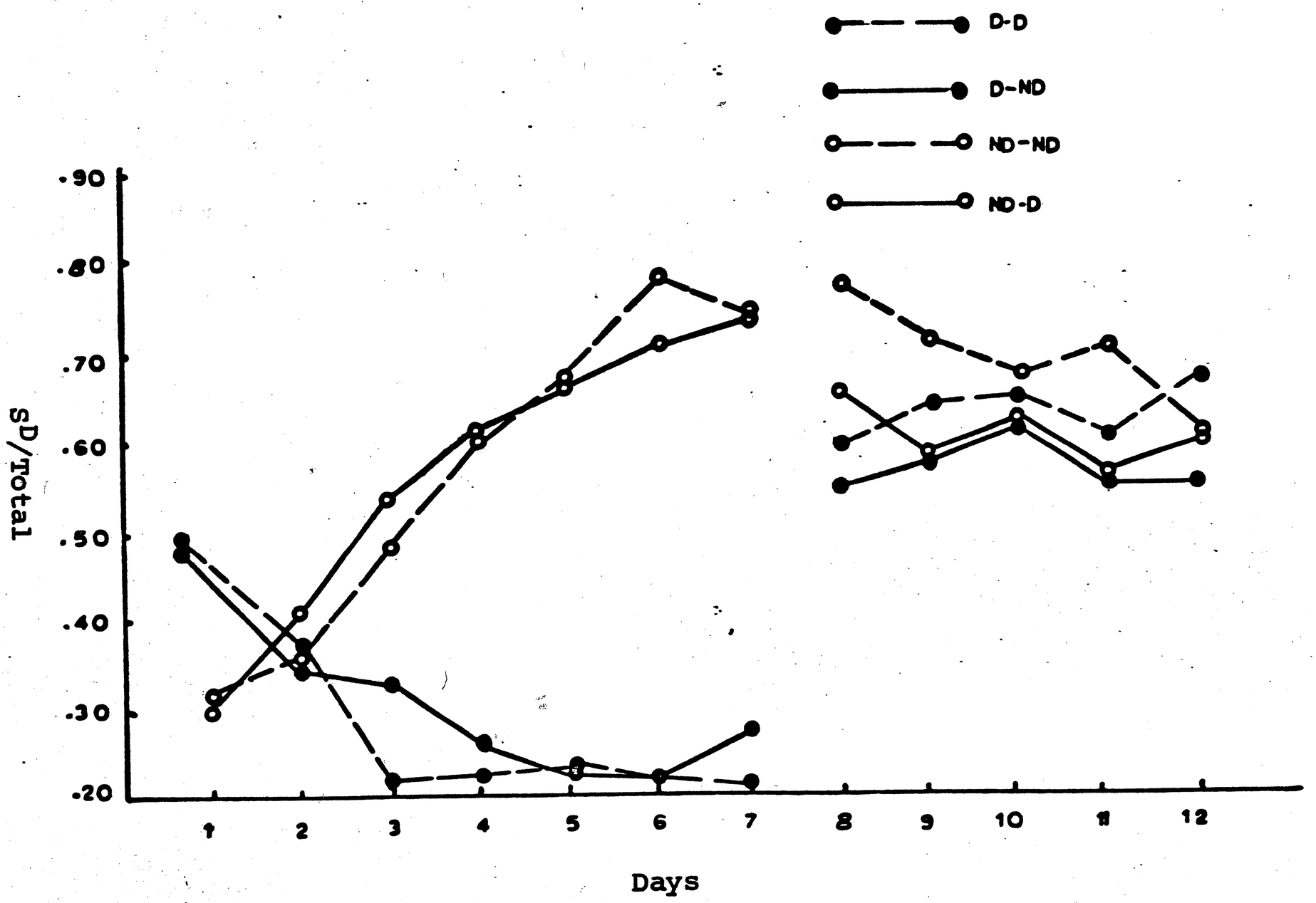
1. This study was published after present one was initiated.

in that it is more parsimonious than the latter, these two possible explanations are not mutually exclusive. If, however, one refers to Figure 6, which represents the proportion of S^D to total responses per day of acquisition and extinction for the four acquisition-extinction drug combinations, it is clear that while the D Ss differed from the ND Ss in acquisition, this difference appears to dissipate greatly during extinction. While D Ss never reached the 50% level during acquisition, by the first day of extinction these Ss were above 50% level. However, it should be noted that this increase in the S^D/total measure appears less meaningful if one observes the initially high S^D/total measure for the drugged Ss on the first 2 days of acquisition. This initial high ratio, however, (approaching 45%) seems to be an artifact of the experimental situation. More specifically, since all Ss were trained to bar press in a ND condition when acquisition training began under the D condition state-dependent learning should be active. This situation appears to have caused a sharp decrement in responding and since the first acquisition trial was always an S^D trial, the S^D/total measure of those Ss were raised abnormally. If, for example, a D S responded only twice on the first day of acquisition due to the above-mentioned ND to D shift and the first period was an S^D period, his S^D/total ratio would in all probability be 100%. The ratio although high, of course, does not indicate that a S learned a tone, no-tone discrimination.

There are, however, two biasing factors which present some problems for the above interpretation. As was stated earlier there were always 16 S^D periods and 14 S^Δ periods, and an S^D period was always the first on any given day. The first biasing factor raises the chance value to 53% while the second biasing factor (S^D period first) cannot be calculated with available data.

Figure 6. SD responses/SD total per day


D-D
D-ND
ND-ND
ND-D
.90
.80
.70
.60
.50
.40
.30
.20
SD/Total
1
2
3
4
5
6
7
8
9
10
11
12
Days

It, therefore, is possible that non-drug Ss learned something either qualitatively or quantitatively different than drugged Ss, resulting in differential state change effects when going from drugged or non-drugged acquisition. An interpretation of this sort has, in fact, been presented by Sachs (1967) and is consistent with the present data (Refer to Figures 4 and 5.).since a differentiation was found between the ND-D and D-ND groups. However, again referring to Figure 5, both the ND-ND and D-D groups appear to have parallel generalization gradients indicating identical functions, even though they had different acquisition functions. It, therefore, may be concluded that since the experimental task was a relatively easy one extended over seven days it is likely that drugged and non-drugged Ss abstract similar information from acquisition even though it is not possible to confirm this possibility at this time since the second biasing factor cannot be calculated.

It is, therefore, concluded with the above evidence that the D and ND Ss differed from one another in acquisition mainly as a function of performance variables bringing about this differentiation.

With this learning performance distinction tentatively established, the four acquisition-extinction drug combinations (Acquisition x Extinction x Frequency interactions) may now be compared. Referring to Figures 4 and 5, it is apparent that there are differences in the height of the four functions, The differences are generally ordered in the following manner (from most responses to least): D-D, D-ND, ND-ND, and ND-D. It appears that the first two groups responded most frequently in extinction due to the previously established food x drug interaction. When, for example, a D S, whose responding was suppressed by the food x drug interaction, was placed in extinction the above suppression was removed. This removal can be looked at or defined as disinhibition, and as with many disinhibitory phenomena when an inhibitory

influence is removed, responding may increase in greater amounts than expected (this phenomenon may be thought of as postinhibitory rebound). It should also be mentioned that the D-ND group should be expected to have a smaller postinhibitory rebound effect than the D-D group since only in the former group is the release from inhibition accompanied by a change in state, where dissociation effects should, of course, be active. Aside from the above difference in height among the 4 acquisition-extinction drug combinations, there is also a difference in their generalization functions. Referring to Figures 4 and 5, once again, it is clear that the ND-D function is flatter than the remaining 3 functions. This differentiation along with Figure 3 (which represents a significant acquisition-extinction interaction) indicates that different processes may mediate D to ND and ND to D dissociation. Since Figure 3 indicates dissociation in both the ND to D and D to ND direction (ND-D $<$ ND-ND and D-ND $<$ D-D) the differentiation between the generalization function of the ND to D and D to ND implies a dual dissociation mechanism. A tentative explanation is that for the D to ND group the removal of the drug in extinction has the effect of the removal of one set of discriminative stimuli. This effect, of course, would not upset the generalization gradient since the perceptual system quite possibly remains unaffected due to the fact that the S is returning to a normal, non-drugged state. When, however, the drug condition is added to the previous non-drug acquisition condition, there is not the removal of a set of discriminative stimuli but the addition of a new and unusual state, a state which could change or disrupt perceptions of the Ss. This hypothesis is supported by the finding that only for the ND to D group was a generalization gradient not observed. Additional support for this hypothesis is found in the phenomenon of asymmetrical drug-induced dissociation -

in other words, if two qualitatively different types of dissociation could be identified, two underlying mechanisms could be postulated to account for those possible types of dissociation. Overton (1968), in fact, reports that training often appears to transfer more completely in the ND to D than the D to ND direction. While this view is supported by Figure 3 (difference between the ND to ND and ND-D smaller than difference between the D to D and D to ND), a note of caution should be added. Since stimulus generalization would appear to be a more sensitive measure of possible perceptual effects acting during dissociation, the observation of a flattened generalization gradient for the ND to D group indicates less transfer or greater dissociation than the D to ND group where a non-flattened generalization gradient is found. The flattened ND to D gradient can, thus, be accounted by the S's perceptions of the auditory stimulations being altered. This alteration could result in the stimuli being perceived as qualitatively different than the original CS, so different that the auditory stimuli are now part of a different generalization continuum - hence a flattened generalization gradient. To conclude, then, it appears that 2 different types of dissociation were observed in the present study: discriminative stimuli mediated dissociation (the D to ND group) and perceptual mediated dissociation (the ND to D group). It, thus, appears that perceptual changes play at least a part in the phenomenon of dissociated learning. It should be stressed, however, that the previously mentioned differentiation between discriminative stimulus and perceptually mediated dissociation is not the sole interpretation of the presented experimental data. The interpretations, therefore, should be viewed as tentative theoretical views consistent with the experimental data which await further experimental verification.

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VITA

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